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Structural proteins, NSs virulence factors and pathogenesis of Rift Valley Fever Virus in infected hosts

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Abstract

The Rift Valley Fever (RVF) is an emerging zoonotic disease caused by the RVFV of the family *Bunyaviridae* and the genus *Phlebovirus*. The RNA genome of the RVFV is composed of three single-stranded segments (S, M, and L) that are negative or ambisense and have virions that range in size from 90 to 110 nm. Since RVFV is an RNA virus, it protects its genomes by providing either a protein shell or a protein coat for their genomes. Apoptosis is the other defensive mechanism that removes infected cells and limits viral reproduction and dissemination on behalf of hosts. The NSs protein, which inhibits the mammalian type I interferon system, is the main virulence factor for RVFV infection and interacts with other molecules on various levels. In general, ruminant immunization is the most effective strategy for preventing human disease due to domestic ruminants' involvement in the epidemiological cycle and the fact that humans typically become infected after coming into contact with viraemic animals. Therefore, research that will provide a more thorough understanding of the remaining viral structural and non-structural proteins and their molecular basis, which are accountable for its pathogenicity is advised for the feature.

Keywords: genome, pathogenesis, rift valley fever virus, structural proteins, virulence factors

1. Introduction

The Rift Valley Fever Virus (RVFV) is a serious veterinary and public health threat that has resulted in widespread disease outbreaks in livestock and people in the majority of African nations as well as in the Arabian Peninsula since 2000, particularly in Yemen and Saudi Arabia (Flick & Bouloy, 2005; Pepin *et al*., 2010). The virus was first discovered by Daubney and Hudson in Kenya's Great Rift Valley in 1930 (Daubney & Hudson, 1931; Peng & Lozach, 2021). The RVFV is a member of the family *Phenuiviridae* in the order *Bunyavirales* and the *Phlebovirus* genus of RNA viruses, which are transmitted by mosquitoes (Abudurexiti *et al*., 2019). *Bunyaviruses* have an enveloped, trisegmented, negative-sense, and a single-stranded RNA genome with a negative orientation (Ahmed *et al*., 2020) that replicates in the cytosol

(Lozach *et al*., 2010). These *Bunyaviruses* are distinguished by the fact that viral particle assembly occurs in the Golgi complex inside cisternae, where virions have been demonstrated to bud (Schmaljohn & Nichol, 2007). The viral antigen was also detected by immunohistochemistry (IHC) in different cells such as the liver, spleen, lymph nodes, kidneys, and lungs (Shieh *et al*., 2010), whereasthe antibodies that can neutralize viruses are produced in response to the expression of the glycoproteins Gn and Gc by recombinant MVAs (Calvo-Pinilla *et al*., 2020).

The Rift Valley Fever (RVF) is caused by the RVFV, which spreads over a significant portion of the nation following heavy rains and prolonged flooding because these weather conditions are favorable for the virus' vectors. Aedes mosquitoes from floodwaters, such as *Ae.mcintoshi* or *Ae.vexans*, act as vectors in areas where the virus is prevalent, and they may transovarially transfer the disease to their young (Ikegami & Makino, 2011). Different mammalian species are infected by the virus, whereas young animals are far more vulnerable and much more likely to perish. Lambs are "very sensitive," calves are "highly susceptible," and humans are "moderately susceptible" to the sickness. Although the clinical symptoms of RVF are typically nonspecific, high rates of mortality in young animals, high rates of abortion, and flu-like symptoms in humans are suggestive (Knight-Jones *et al*., 2014). Particularly, the susceptibility to RVFV varies with age, with mortality rates typically being 90% or higher in young lambs and 20% to 30% in older lambs and ewes. Abortion in ewes is frequently the only evidence of infection in a flock, and adult sheep frequently die suddenly without exhibiting any clear symptoms (Odendaal *et al*., 2019). In general, the disease is most common in newborns and pregnant animals, causing a high rate of mortality and abortion and causing significant economic problems in endemic countries (Chevalier *et al*., 2010; Carnec *et al*., 2014).

Additionally, the livestock trade contributes to the spread of the disease into disease-free areas, thereby expanding the geographical distribution of RVF (Chevalier *et al*., 2010). As a result, the RVF has caused substantial outbreaks in a number of Sub-Saharan African countries, including Kenya, Tanzania, Somalia, South Africa, Madagascar, Egypt, Sudan, Mauritania, Senegal, Saudi Arabia, and Yemen, making these countries almost in danger. A lack of appropriate biosafety procedures was also one of the contributing factors to the spread of RVF in pandemic areas between infected animals and humans (Ikegami & Makino, 2011). Therefore, the objective of this paper was to review the structural proteins, NSs virulence factors, and pathogenesis of the RVFV in infected susceptibilityhosts.

2. Literature review

2.1. Structural proteins and RVFV virulence factors

2.1.1. Structural proteins of the RVFV

The RNA genome of the RVFV is composed of three single-stranded segments (designated by the letters S, M, and L) that are negative or ambisense and have virions that range in size from 90 to 110 nm in diameter (Calvo-Pinilla *et al*., 2020). The nucleocapsid protein (N) and non-structural S (NSs) genes are encoded by the S-segment in an ambisense fashion. Whereas the Gn and Gc genes, NSm (NSm2) and 78 kDa (NSm1), are encoded by the M-segment, and the L-segments encode the L-RNA-dependent segment's RNA polymerase (L) gene (Ikegami & Makino, 2011), as the RVFV viral structural proteins are depicted in **figure 1** below. NSm and NSs, two nonstructural proteins, are expressed during infection (Calvo-Pinilla *et al*., 2020), and both of them are not incorporated into viral virions (Struthers & Swanepoel, 1982). The Gn and Gc carboxy-terminus contain a Golgi targeting signal and an endoplasmic reticulum (ER) retrieval signal, respectively (Carnec *et al*., 2014).

The S-segment codes for two proteins with opposite polarities: the5 half of the genomic and antigenomic RNA encoding the non-structural protein, NSs, and the N nucleoprotein, respectively (Carnec*et al*., 2014).The N protein of RVFV is a 27-kDa protein that is essential for virus assembly through interactions with the viral envelope glycoproteins (Gn and Gn) (Overby*et al*.,2007a; Gaudreault *et al*., 2019). By concentrating on the IFN effector PKR, NSs also block the IFN response. PKR is degraded by proteasomes as a result of NSs, as this protein would otherwise be triggered by viral RNA and prevent RVFV replication (Ikegami *et al*., 2009; Kainulainen *et al*., 2016).

The M-segments code for a polyprotein precursor, which is cleaved in the ER to generate both Gn and Gc envelope glycoproteins. Processing of the polyprotein also gives rise to two non-structural proteins, NSm1 and NSm2 (Suzich*et al*., 1990; Gerrard & Nichol, 2007). The formation of the envelope glycoproteins Gn and Gc in the Golgi apparatus, together with the subsequent recruitment of the nucleoprotein N to the same area, signals the beginning of this process (viral budding). Since bunyaviruses don't express any matrix proteins, N is thought to be recruited towards the Golgi by its contact with the cytosolic tails of the envelope glycoproteins. This allows ribonucleoprotein (RNP) encapsidations during particle formation (Overby *et al*., 2007a; Strandin *et al*., 2011). The Gn-Gc heterodimers that make up the viral spikes are targeted and retained in the Golgi by the cytosolic tail of Gn (Shi *et al*., 2007; Carnec *et al*., 2014).

In reality, during co-translational processing of the polyprotein encoded by the M-segment, the Gn and Gc glycoproteins shift from the ER to the Golgi because the Gn C-terminal region often carries a Golgi targeting signal. The Gc, on the other hand, persists in the ER when expressed independently (Matsuoka *et al*., 1994; Gerrard & Nichol, 2002; Carnec *et al*., 2014).A basic di-lysine motif (KKXX-COOH), conserved among phleboviruses and found in their short C-terminal domain of Gc, may account for its ER retention in the absence of Gn. This ER retention led to the Gc being transported to the Golgi only upon interaction, with Gn acting as a chaperone in this process (Overby *et al*., 2007b; Carnec *et al*., 2014). The L-segments code for the RNA-dependent RNA polymerase (Müller*et al*., 1994; Carnec*et al*., 2014).

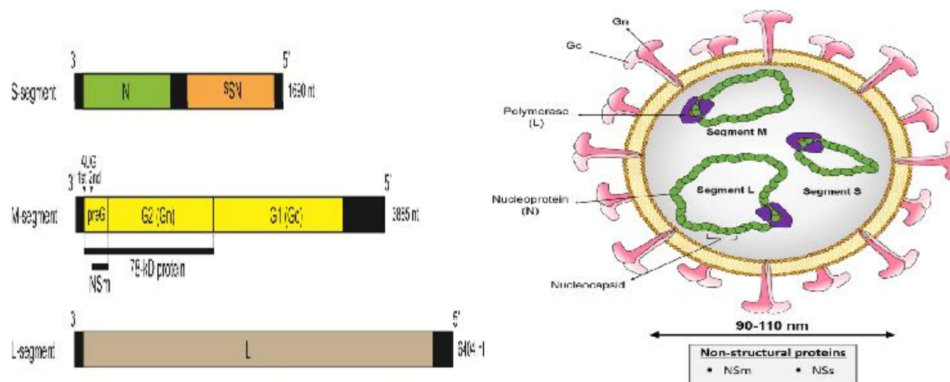

Figure 1:Schematic diagram of RVFV viral structural proteins
(Ikegami & Makino, 2011; Calvo-Pinilla *et al*., 2020)

2.1.2. NSs viral virulence factors

Several RNA viruses protect their genomes in one of two ways: by providing either a protein shell or a protein coat for their genomes. This mechanism is called encapsidation, in which the process takes place in a variety of ways, both functionally and structurally. As a result, most of those negative-sense RNA viruses' encapsidatestheir genomes by coating the length of their RNA with a nucleocapsid protein (N). Even though capsid and N all bind RNA, the resulting RNA-protein complexes differ, and it is not possible to make generalizations about the proteins involved in the encapsidation process across entirely RNA viral families (Raymond *et al*., 2010).

One of the host defensive mechanisms that removes infected cells and limits viral reproduction and dissemination is the induction of apoptosis in virus-infected cells and subsequent phagocytosis of these cells (Nainu*et al*., 2017). RVFV has been shown to cause apoptosis mostly by activation of caspase-8. However, the RVFV NSm protein inhibits apoptosis in target cells and caspase-8 production in infected cells, delaying apoptosis and enabling the effective release of RVFV progeny within the first 24 hours following infection (Won *et al*., 2007). As a result, the majority of the progeny virus is released prior to virus-induced apoptosis. RVFV replicates poorly in mouse macrophages and has lower pathogenicity in mice when lacking the NSm protein, showing that NSm plays a role in viral replication (Kreher *et al*., 2014).

The NSs protein, which inhibits the mammalian type I interferon (IFN) system, functions as the main virulence factor for RVFV infection, and it interacts with other molecules on various levels (Kainulainen *et al*., 2016). Additionally, the NSs were previously characterized as an effective IFN induction inhibitor that interferes with RNAP II action in the host cell (Billecocq *et al*., 2004). NSs work by attracting the transcriptional repressor Sin3A-associated protein (SAP30) to the IFN promoter (Le May *et al*., 2008; Kainulainen *et al*., 2016) and by impairing the general transcription factor TFIIH by sequestering the p44 subunit and destroying the p62 subunit via proteasomal degradation (Pichlmair*et al*., 2012; Kainulainen *et al*., 2016). In general, the S-segment uses an ambisense approach to express the nucleoprotein N and non-structural protein NSs, both of which are major virulence factors, particularly the NSs (Kreher *et al*., 2014).

2.2. Pathogenesis

It has been demonstrated that low pH alters the biochemical characteristics of viral glycoproteins and triggers fusion of cells expressing RVFV glycoproteins, which suggests that host cell entry of phleboviruses is dependent on low pH (Lozach *et al*., 2010). After binding to a cellular receptor that has not been identified, RVFV virions enter the host cells in a pH-dependent manner (*Filone et al., 2006*), which is most likely mediated by a clathrin-mediated endocytic pathway (Lozach *et al*., 2010). Then viral uncoating is followed by the release of the viral ribonucleocapsid (RNP), which is made up of viral genomic RNA segments and N protein. After that, primary transcription is carried out by the viral polymerase, which is probably connected to the RNP, to produce viral mRNA (Ikegami and Makino, 2011). As a result, the pathogenesis of RVFV in sheep may be significantly influenced by direct infection and damage to endothelial cells, as well as increased activation of coagulation caused by consumption of platelets and coagulation factors (Odendaal *et al*., 2019).

Viral RNA replication begins 1–2 h after infection (Ikegami *et al*., 2005), and an increase in the amount of viral genomic RNA results in increases in viral mRNAs and proteins. Similar to other bunyaviruses, the RNP is likely packaged into viral virions by its interaction with the cytoplasmic domains of Gn and Gc at the Golgi apparatus (Overby *et al*., 2007a).The co-packaging of the M and S-segments supported the packaging of the L-segment, suggesting that the three distinct RNA segments could be co-packaged in a coordinated manner (Terasaki *et al*., 2011). The encapsidation process plays a vital

role in multiple steps within the replicative cycle, including transcription and replication by the RNA-directed RNA polymerase (RdRp) and packaging of the genome into virions (Schmaljohn & Hooper, 2007). Then, the 122 glycoprotein capsomers that make up the RVFV virion surface form a highly symmetric T = 12 icosahedral lattice (Freiberg *et al*., 2008), which is most likely composed of 720 Gn-Gc heterodimers (Huiskonen *et al*., 2009). Smith *et al*. (2019) reported that the new progeny's propagation was described in various cell cultures after it had matured.

2.3. Prevention and control of RVF

Due to domestic ruminants' involvement in the epidemiological cycle and the fact that humans typically become infected after coming into contact with viraemic animals, ruminant immunization is the most effective strategy for preventing human disease. Live and inactivated vaccines are both available for livestock (Chevalier *et al*., 2010; LaBeaud *et al*., 2010). The use of bed nets and insect repellents during outbreaks is also advised, as is the implementation of information campaigns for those who may be at risk (farmers, veterinarians, butchers, employees of slaughterhouses, etc.), the banning of the slaughter and butchering of ruminants during epizootics, and the proper disposal of dead animals (Chevalier *et al*., 2010). In general, the effective countermeasures must be developed, and surveillance and diagnostic tools must be used. Furthermore, the introduction of RVFV would have serious long-term negative consequences for the healthcare, agriculture, and travel economic sectors (Mandell & Flick, 2011).

3. Conclusion and Recommendations

The RVFV is a serious veterinary and public health threat that has caused widespread disease outbreaks in livestock and people in various African countries and the Arabian Peninsula since 2000. RVF is an emerging zoonotic disease caused by the RVFV of the family *Bunyaviridae* and the genus *Phlebovirus*, spread by mosquitoes and causing a high rate of mortality, abortions, and economic problems. Since RVFV is an RNA virus, it protects its genomes by providing either a protein shell or a protein coat for their genomes. While the NSs protein is the primary virulence factor for RVFV infection, apoptosis is a protective mechanism that eliminates infected cells and restricts viral reproduction and spread. In general, ruminant immunization is the most effective strategy for preventing human disease due to domestic ruminants' involvement in the epidemiological cycle and the fact that humans typically become infected after coming into contact with viraemic animals and their products at all. Additionally, research that will provide a more thorough understanding of the remaining viral structural and non-structural proteins and their molecular basis, which are accountable for the pathogenicity of RVFV, is advised for the feature.

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